IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

In re Patent Application of

MALISSEN et al — Atty. Ref.: 3665-113; Confirmation No. 9194

Appl. No. 10/502,332 — TC/A.U. 1632

Filed: July 23, 2004 — Examiner: TON

For: MUTATED GENE CODING FOR A LAT PROTEIN AND THE BIOLOGICAL APPLICATIONS THEREOF

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May 22, 2006

Commissioner for Patents
P.O. Box 1450
Alexandria, VA 22313-1450

Sir:

RESPONSE

Responsive to the Official Action dated April 20, 2006, the applicants elect, with traverse, the subject matter of the Examiner's Group I.

Reconsideration and withdrawal of the restriction requirement are requested.

At a minimum, the Examiner is requested to withdraw the restriction requirement in so far as the Examiner asserts that the subject matter of the Examiner's Groups I and II fail to share the same or corresponding special technical feature.

The applicants submit that the subject matter of the Examiner's Groups I and. II are linked by the same inventive concept, i.e., that the mutant LAT protein leads to exaggerated TH2 cell differentiation.

The Examiner is understood to believe that this feature fails to define a contribution over the prior art, because mice having mutated LAT genes are known from .Sommers et al. (J. Exp Med 194(2): 135-142 (July 16, 2001)) where the authors allegedly teach mice which contain a mutation of the distal four tyrosines of LAT and because LAT is further known from Zhang et al (Immunity 10: 323-332 March 1999) for its role in T cell development.

The applicants respectfully disagree with the Examiner's interpretation of the art and claims. Specifically, the Examiner is requested to see the abstract of Sommers et al., for example, which stipulates that the aim and conditions of the study are as follows:

> "In this study, we (the authors] examined whether these four tyrosine residues were essential for T cell development by generating LAT "knock-in" mutant mice that express the 4YF mutant protein under the control of endogenous LAT regulatory sequences. .... development was arrested at the immature CD4-CD8- stage and no mature T cells were present." See Abstract.

The mutation of the four distal tyrosines thus leads to the arrest of thymocyte development in Sommers et al. while, on the contrary, the presently claimed invention provides a non-human animal having a mutated LAT gene coding for a mutant LAT protein, wherein the mutant LAT protein leads to an exaggerated TH2 cell differentiation

Therefore the claimed animals, cells and genes define over the cited art and share the same or corresponding special technical feature which defines a contribution over the art of record.

Withdrawal of the restriction requirement and examination of all of the claimed subject matter are requested.

At a minimum, the Examiner is requested to examine the subject matter of the Examiner's Groups I and II together.

Rejoinder and allowance of an claims defining methods of making and/or using products defined by allowable claims are requested at an appropriate time.

Respectfully submitted,

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